Mechanisms of Biotic Interactions
Hailong Guo - Poster-B118

Abstract Title: DISTINCT MODES OF DEREPRESSION OF AN ARABIDOPSIS IMMUNE RECEPTOR COMPLEX BY TWO DIFFERENT EFFECTORS

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Abstract

Plants intracellular nucleotide-binding leucine-rich repeat (NLR) immune receptors are encoded by Resistance (R) genes and confer specific resistance to pathogen races that carry the corresponding recognized virulence factors known as effectors. RRS1 and RPS4 are a pair of NLR proteins that function together to recognize two bacterial effectors, PopP2 and AvrRps4. The RRS1-R allel e in Arabidopsis accessions Nd-1 and Ws-2 confers AvrRps4 and PopP2 recognition, whereas Col-0 allele of RRS1 (RRS1-S) confers AvrRps4, but not Pop P2, recognition. We previously reported that RRS1-R/RPS4 recognizes AvrRps4 and PopP2 via an integrated WRKY domain in RRS1-R that mimics the effector's authentic targets. Here we show that the inactive conformation of RRS1 is held together by WRKY interactions with an adjacent RRS1 domain prior to effector detection. AvrRps4 interactions with the WRKY domain disrupt its negative regulation of the complex. While PopP2 de-represses the complex via inter-domain reconfigurations involving the phosphorylation of the WRKY domain (DOM5) and its neighboring Domain 6 (DOM5+6) of RRS1-R. Intriguingly, DOM5+6 of RRS1-R, but not RRS1-S, is phosphorylated. Five phosphorylated Ser residues of RRS1-R were identified using immunoprecipitation and mass spectrometry (IP-MS) and combined mutations of all five DOM56 phosphorylation sites abolished PopP2, but not AvrRps4, recognition, suggesting AvrRps4 and PopP2 have different modes of action to de-repress the complex.

Mechanisms of Biotic Interactions
Sebastien Ribeiro - Poster-B121

Abstract Title: DELETION OF CASSIICOLIN-ENCODING GENE CAS1 FROM CORYNESPORA CASSIICOLA CAUSES A LOSS IN VIRULENCE ON RUBBER TREE

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Abstract

Corynespora Leaf Fall is an important disease affecting rubber tree in Asia and Africa, caused by the necrotrophic fungus Corynespora cassiicola . A small protein toxin, the cassiicolin, is suspected to play

an important role in the early stages of the disease. To demonstrate its role and evaluate the possible involvement of other effectors, a deletion mutant targeting the cassiicolin gene Cas1 was created from the highly aggressive isolate CCP. Wild-type and deletion mutant were not found different in terms of mycelium growth, sporulation and germination. Deletion of the Cas1 gene induced a complete loss of virulence on two susceptible rubber clones, as demonstrated by controlled inoculations on non-detached leaves. However, the mutant strain conserved some residual virulence when inoculation was conducted on detached leaves, notably with longer incubation times or when the leaves were wounded. The average filtrate toxicity analyzed on a range of clones was found reduced in the mutant compared to the wild-type, but not suppressed. Our results demonstrate: 1) that cassiicolin is indeed a necrotrophic effector conferring virulence to the CCP isolate in specific rubber clones, and 2) that other effectors potentially associated with saprotrophy are responsible for the symptoms observed in senescing/wounded tissues.

Mechanisms of Biotic Interactions

Fabienne Micheli - Poster-B188

Abstract Title: IN SILICO ANALYSIS OF THE TcPR-10MUT TRANSPORT MECHANISM IN FUNGI

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Abstract

Pathogenesis related-proteins 10 (PR-10) have ribonuclease and antifungal activity. A PR-10 from Theobroma cacao (TcPR-10) identified in a cDNA library from the cacao- Moniliophthora perniciosa interaction showed antifungal activity against M. perniciosa and Saccharomyces cerevisiae , and the TcPR-10 was internalized by both organisms. Previous studies indicated that the antifungal activity and internalization of TcPR-10 appears to be related to Snq2 membrane permeates and transporters. In order to investigate the antifungal action of TcPR-10 and its internalization in fungal cells, molecular docking was performed to check the interaction between the ABC (Snq2) transporter and TcPR-10 mut, recombinant mutant protein with reduced allergenicity potential. The three-dimensional structure of TcPR-10 mut and Snq2 were modelled and used in molecular docking analysis using the ClusPro online toolkit. Snq2 interacted with TcPR-10 mut in its transmembrane domain, suggesting the internalization of this protein by means of this transporter, similarly to the transport of alkaloids.

Mechanisms of Biotic Interactions

Qin He - Poster-B192

Abstract Title: A PLANT PATHOGEN EFFECTOR UTILIZES HOST SUSCEPTIBILITY FACTOR NRL1 TO DEGRADE THE IMMUNE REGULATOR SWAP70

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